de BCVC/CVC/1.000 días, mortalidad y hospitalización por BCVC) comparado a pacientes con sellado con heparina[3]. La resistencia bacteriana a G ha sido objetivada[4], pero nuestra experiencia desde julio de 2003 en pacientes con CVC que ingresan en la unidad y se tratan con sellado de G con dosis más bajas (dato que consideramos fundamental por su influencia en la iatrogenia) que las administradas en otras unidades no objetiva resistencia bacteriana ni ototoxicidad después de 9 años de evolución[3]. Al observar nuestros resultados hay que señalar lo publicado por Beathar y Urbanes[1] cuando hacen referencia al valor de la calidad asistencial de una unidad de HD según el número de BCVC/CVC/1.000 días que obtiene cuando se aplica la asepsia universal, el resultado es excelente cuando el valor es ≤ 1. En nuestro caso, la práctica de la asepsia universal + la profilaxis consigue que el número de BCVC/CVC/1.000 días sea de 0,17. Aunque sin poder comparar con ningún estudio, en 9 años conseguir una mortalidad, retirada de CVC y hospitalización por BCVC del 0,8, 2,4, y 3,2%, respectivamente es un dato estimable conseguido por la profilaxis con G + asepsia universal, subrayando además la ausencia de endocarditis y espondilodiscitis, a excepción del único paciente fallecido por sepsis. La asepsia universal estricta[5] en cualquier procedimiento de manejo del CVC es inseparable de la profilaxis para disminuir la morbimortalidad infecciosa bacteriana asociada a la BCVC.

Conclusiones. Este estudio prospectivo observacional de 9 años de duración sobre 126 pacientes en HD con CVC objetiva: *1)* La profilaxis con sellado intraluminal de las ramas del CVC con G no causa resistencia bacteriana a gérmenes sensibles al antibiótico; *2)* No diagnosticamos ototoxicidad clínica, y *3)* La profilaxis con dosis baja de G administrada (comparada a mayor dosis de otras investigaciones[3] puede influir en que no aparezcan resistencia ni ototoxicidad.

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The relationship between serum and urine NGAL and graft function in pediatric renal transplant recipients

Relación entre niveles de NGAL en suero y orina y función del injerto en pacientes pediátricos trasplantados renales

Dear Editor,

We previously serially measured the serum and urine neutrophil gelatinase associated lipocalin (NGAL) during the first week after renal transplantation and found that mild ischemic injury may increase this lipocalin.[1] In this study, we investigated the level of this marker after one month post-transplant with the aim of finding a relationship between NGAL quantities and graft function.

Twenty-one pediatric renal transplant recipients without any infection at the time of assessment were included in this study. Glomerular filtration rate was estimated by Schwartz equation and DTPA scan concurrently. Scintigraphic measurement of GFR was performed using an ADAC single-headed gamma camera with the following formula:

$$\text{Total renal uptake percent}\,(\%) = (k - b)/e - \mu x$$

Pre-post – k: kidney count; b: background count; x: renal depth; e: constant; μ: attenuation coefficient of 99mTc in soft tissue ($0.153\,cm^{-1}$); GFR = total renal uptake percent (%) $\times$ 100 $\times$ 9.81270 – 6.82519.

The mean age of patients was 9.9 ± 3 years old. Nine patients (43%) were male. The mean time from transplantation was 6.8 ± 2.47 years. The mean serum creatinine was 1.16 ± 0.18 mg/dl. The mean Schwartz calculated GFR was 69.8 ± 12.2 cc/min/1.73 m^2. The mean DTPA measured GFR was 50.6 ± 16 cc/min/1.73 m^2. All patients had GFR less than 90 cc/min/1.73 m^2 by scan and Schwartz formula. The mean serum NGAL was 140 ± 94 ng/ml (15–324 ng/ml). The mean urine NGAL was 17.8 ng/ml (3.2–68 ng/ml).

We assessed the correlation between serum NGAL and serum creatinine, Schwartz GFR, and DTPA-related GFR. The coefficient of correlation with serum creatinine was 0.67 ($P = 0.09$), −0.2 ($P = 0.3$) with Schwartz GFR, and −0.26 ($P = 0.46$) with DTPA GFR. Regarding urine NGAL, the correlation coefficient with serum creatinine was 0.2 ($P = 0.37$), −0.007 ($P = 0.9$) with Schwartz GFR, and −0.24 ($P = 0.48$) with DTPA GFR.

We did not find any significant association between the transplant time and serum NGAL ($r = 0.05$, $P = 0.8$) and urine NGAL ($r = 0.06$, $P = 0.77$). Three patients had slow graft function in this study without need for dialysis in the first week post-transplant. The mean serum and urine NGAL was not different between patients with SGF and those without SGF (for serum NGAL 106 vs 145.5 ng/ml and for urine NGAL 12.2 vs 21.6 ng/ml).

Studies have shown that expression of NGAL protein is significantly increased during ischemic insults in renal transplant recipients with delayed graft function.[2] Magnusson et al. have shown that plasma NGAL levels were significantly higher than normal in renal transplant recipients.[3] Malyszko et al. also found a strong correlation between serum NGAL and serum creatinine in 100 kidney transplant recipients.[4]

This study is the first study in pediatric renal transplant recipients in which the association between serum and urine NGAL with graft function was assessed long term. We did not find any significant association between the amounts of NGAL in serum and urine with serum creatinine and GFR estimated by Schwartz formula or measured by DTPA scan. We think we cannot use serum and urine NGAL as markers of graft function in pediatric renal transplant recipients, but this result needs confirmation by more studies with more cases.

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